U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 15 January 2003

CASE NO. 2002-BLA-46

In the Matter of

IRENE G. KEILMAN, Survivor of WILBERT A. KEILMAN, SR.
Claimant

v.

BETHENERGY MINES, INC.
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

APPEARANCES:

Heath M. Long, Esquire
For the Claimant

John J. Bagnato, Esquire
For the Employer

Before: RICHARD A. MORGAN
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits filed by Irene G. Keilman, the surviving spouse of Wilbert A. Keilman, Sr., a now deceased coal miner, under the Black Lung Benefits

Act, 30 U.S.C.§ 901, *et seq.* Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.[1]

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on May 13, 2002 in Pittsburgh, Pennsylvania. At that time, all parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations issued. The record consists of the hearing transcript, Director's Exhibits 1 through 39 (DX 1-39), Claimant's Exhibits 1 and 2 (CX 1-2), and Employer's Exhibits 1 through 4 (EX 1-4). Pursuant to leave granted at the formal hearing, the record was held open until June 30, 2002 for the submission of post-hearing briefs (TR 20-21).

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented. Where pertinent, I have made credibility determinations concerning the evidence.

Procedural History

On August 27, 1987, Wilbert Keilman, a former coal miner, passed away (DX 7). On June 9, 2000, the Claimant, Irene G. Keilman, filed the current application for black lung benefits under the Act, as his surviving spouse (DX 1). The claim was denied by the District Director's office on October 12, 2000 (DX 15) and August 2, 2001 (DX 34), respectively. Following Claimant's timely requests for a formal hearing (DX 16, 35), this matter was referred to the Office of Administrative Law Judges for *de novo* adjudication (DX 38, 39). As stated above, a formal

[1] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001 and were to apply to both pending and newly filed cases. The new Part 725 regulations also became effective on January 19, 2001. Some of the new procedural aspects of the Part 725 regulations, however, were to apply only to claims filed on or after January 19, 2001, *not* to pending cases. The Amendments to the Part 718 and 725 regulations were challenged in a lawsuit filed in the United States District Court for the District of Columbia in *National Mining Association v. Chao*, No. 1:00CV03086 (EGS). On February 9, 2001, the District Court issued a Preliminary Injunction Order which enjoined the application of the Amendments except where the adjudicator, after briefing by the parties to the pending claim, determines that the regulations at issue in the instant lawsuit will not affect the outcome of the case. On August 9, 2001, the United States District Court for the District of Columbia issued a decision granting the U.S. Department of Labor's motion for summary judgment in *National Mining Association v. Chao*, dissolved the Preliminary Injunction, and upheld the validity of the amended regulations. On appeal, the D.C. Circuit issued its decision in *National Mining Ass'n, et al v. Dep't of Labor*, _____F.3d_____(D.C. Cir. June 14, 2002), which further addressed the validity and application of the revised regulations. With the exception of a few provisions, the Court affirmed the validity of the revised regulations, as well as its retroactive application. Furthermore, the provisions of revised 20 C.F.R. § 718.205(c)(5) regarding pneumoconiosis hastening the miner's death simply codifies existing case law. Moreover, the Court expressly held that the "hastening death rule" set forth in § 718.205(c)(5) is not arbitrary and capricious. Accordingly, under the facts herein, the Amendments do not affect the outcome of this claim.

hearing was held on May 13, 2002, and the record was held open until June 30, 2002 for the submission of written closing argument (TR 20-21).[2]

Issues

The only contested issue, as set forth on the Form CM-1025 transmittal sheet, and confirmed at the formal hearing, is as follows: Whether the miner's death was due to pneumoconiosis? (DX 38; TR 5-6).

Findings of Fact and Conclusions of Law

Background and Employment History

A. Coal Miner

The parties stipulated, and I find, that former miner, Wilbert A. Keilman, Sr., engaged in coal mine employment for at least 33 years (DX 34,38). Furthermore, any discrepancy between the above-stipulated number of years of coal mine employment and the 35 years alleged by the Claimant (TR 8) is inconsequential for the purpose of rendering a decision on the merits.

B. Date of Filing

The Claimant filed her claim for survivor's benefits under the Act, on June 9, 2000 (DX 1). Although the claim was filed almost 13 years after her husband's death, there is no time limit on the filing of a claim by the survivor of a miner. *See* 20 C.F.R. § 725.308. Accordingly, the Employer stipulated, and I find, that the claim was timely filed (DX 34, 38).

C. Responsible Operator

The Employer, BethEnergy Mines, Inc., has stipulated, and I find, that it is the properly designated responsible coal mine operator in this case, under Subpart G, Part 725 of the regulations (DX 34, 38).

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[2] The record also contains the miner's application for benefits, dated January 20, 1982 (DX 37-1), which was denied by the District Director's office on July 27, 1983 (DX 37-12) and June 10, 1986 (DX 37-16). Although the miner filed a timely request for a formal hearing on June 18, 1986 (DX 37-17), he subsequently filed a request to withdraw his claim on the grounds that the State occupational disease benefits which he was receiving exceeded the amount of benefits he would be entitled to, even if he were granted Federal benefits (DX 37-23). Employer stated that it did not object thereto (DX 37-25), and the District Director accepted the withdrawal (DX 37-24). Accordingly, the miner's withdrawn claim is considered not to have been filed. *See* 20 C.F.R. § 725.306. Furthermore, the State award of black lung benefits (DX 37-22) is not controlling herein, because the underlying statutes, regulations, and evidence are not identical with those governing this Federal black lung claim. Moreover, the State finding of disabling pneumoconiosis, in July 1985, does not establish that the miner's death, almost two years later, was due to pneumoconiosis.

D. Dependents

The Claimant, Irene G. Keilman, has no dependents for purposes of augmentation of benefits under the Act (DX 1, 34, 38; TR 8).

E. Personal Background and Other Lay Evidence

The former miner, Wilbert A. Keilman, Sr., was born on January 5, 1930. He married Irene G. Keilman (nee Travis) on September 1, 1951. They remained married until his death on April 27, 1987. Mrs. Keilman (hereinafter "Claimant") has not remarried since her husband's death (DX 1, 6, 7; TR 7-8).

Claimant testified that her late husband, Wilbert A. Keilman, Sr., engaged in coal mine employment for 35 years all in underground mines. He worked in various coal mine jobs, as a "scrapper with his father on a cutting machine," buggy runner, long wall worker, and section foreman. His last coal mine job, as a section foreman, entailed working in "low coal" in dusty conditions. Claimant described the height of the mine as only 2 ½ feet. Mr. Keilman left the coal mines on September 1, 1981, when he suffered a back injury. He was never gainfully employed after his back injury (TR 8-10).

Claimant testified that her husband began having some breathing problems before he left the coal mines, which worsened over a period of time (TR 10, 17). In the 1960's and 1970's, Mr. Keilman had been an active person, and went hunting, fishing, and bowling. Claimant testified, however, that, because of his breathing condition, her husband could not continue to do the foregoing activities as of the late 1970's and early 1980's. Moreover, Claimant stated that Mr. Keilman had difficulty mowing the lawn; and, he "was panting" when he walked the length of the 40' driveway or climbed the cellar steps (TR 10-11). Claimant also testified that her husband's breathing "got a little worse" from 1981 until his death, in 1987, and, that his coughing also worsened over the same period (TR 11-12). She stated that her husband smoked approximately 6 to 8 cigarettes per day, beginning at age 16, and ending when he was in his early 30's (TR 13-15).

Claimant testified that her husband became ill at home in 1987, and was taken to Lee Hospital, in Johnstown. He was initially placed on a ventilator; but "they took the ventilator out and put a trach (sic) in." Mr. Keilman remained in the hospital for 27 days, and, then, passed away (TR 12-13). Claimant stated that the miner did not take medication for heart or breathing conditions. The only medication he took was Darvocet for his back pain. She also stated that her husband was never treated by a cardiologist. Claimant stated that Dr. Wealey performed her husband's back surgery on referral from the miner's, then, family physician, Dr. Luther (TR 12-13). Subsequently, Dr. Tatarko became the miner's family physician in the years immediately preceding Mr. Keilman's death (TR 17; DX 24).

Medical Evidence

The case file contains various chest x-ray interpretations, pulmonary function studies, arterial blood gas tests, and medical opinions, which had previously been submitted in conjunction with the (withdrawn) living miner's claim (DX 37). As summarized in the Employer's Pre-Hearing Report, the x-ray evidence is mixed. Since the x-ray evidence, at most, shows *simple* pneumoconiosis, and the Employer concedes this issue, such evidence adds little to the analysis of the current survivor's claim. The pulmonary function studies and arterial blood gas tests are nonqualifying. However, total disability is not at issue herein. The medical opinions are conflicting. Dr. Klemens found that the miner was totally and permanently disabled due to pneumoconiosis (DX 37-8; DX 37-14). Dr. Pickerill opined that Mr. Keilman has simple coal worker's pneumoconiosis and a mild restrictive defect, but that he still has the pulmonary reserve to perform his last coal mine job (DX 37-13). Dr. Katter also found that the miner has simple coal worker's pneumoconiosis, but he found no pulmonary impairment whatsoever (DX 37-13). However, none of the foregoing opinions directly address the "death due to pneumoconiosis" issue. Accordingly, even assuming the foregoing medical data is deemed part of the record, notwithstanding the withdrawal of the underlying claim (DX 37-23, 37-24), such evidence does not address the crux of this survivor's case. Similarly, the medical records obtained from Dr. Michael Tatarko's office also do not address the "death due to pneumoconiosis" issue (DX 24).

The evidence which is most relevant to the causation issue herein includes: the miner's death certificate (DX 7), and the medical opinions of Drs. Bush (DX 11, 14, 26, 30; EX 1, 3), Perper (DX 10), Crouch (DX 13, 25), Naeye (DX 23), Tomashefski (DX 28, 31), Hurwitz (DX 29; EX 2), Rizkalla (CX 1), and Fino (DX 32; EX 4). The above-listed evidence is summarized chronologically, since the medical opinions set forth in some of the supplemental reports and/or depositions contain analyses of the earlier findings of other physicians.

The miner's death certificate, which was signed by Dr. J. Patel, states that Mr. Keilman died on April 27, 1987, at age 57. The immediate cause of death was reported as Myocardial Infarction. Under the heading - "OTHER SIGNIFICANT CONDITIONS: conditions contributing to death but not related to the immediate cause given in Part 1 (a)" - Dr. Patel listed "Adult Respiratory Distress Syndrome; Chronic Pulmonary Disease" (DX 7). However, the listing of Chronic Pulmonary Disease is ambiguous, since it does not specify whether the condition was due to coal mine employment or some other etiology. More importantly, the death certificate is neither well-documented nor well-reasoned. It is not even clear whether or not Dr. Patel knew that an autopsy was performed and/or whether the autopsy findings were available prior to the completion of the death certificate (DX 7). In view of the foregoing, I accord little weight to the miner's death certificate.

Dr. Stephen T. Bush, who is Board-certified in Anatomic and Clinical Pathology and in Medical Microbiology (EX 3, Deposition Exhibit 2), performed the autopsy of the deceased miner on April 28, 1987 (DX 11). The initial report, dated April 28, 1987, simply listed the following provisional anatomic diagnoses: adult respiratory distress syndrome; recent acute myocardial

infarction; emphysema, moderate; and, peptic ulcers, duodenum (DX 11). However, Dr. Bush provided a far more extensive report, dated June 10, 1987, which included an external description, primary incision information, the gross description of numerous organs, including the heart and lungs, and, notes regarding the microscopic examination. Based upon the foregoing, Dr. Bush set the following final anatomic diagnoses:

> ACUTE MYOCARDIAL INFARCTION, RECENT, EXTENSIVE
> ADULT RESPIRATORY DISTRESS SYNDROME
>
> Emphysema, Moderate
> Coalworkers' Pneumoconiosis, Simple, Mild
>
> Peptic Ulcers, Duodenum
>
> Acute Tubular Necrosis of Kidneys

(DX 11). In addition, Dr. Bush set forth the following Clinical-Pathological Correlation:

> This patient died with a recent massive acute myocardial infarction with acute respiratory distress syndrome manifested by organizing edema of the lungs diffusely. Pre-existing emphysema is evident. Mild simple coalworkers' pneumoconiosis is noted.

(DX 11).

Dr. Bush issued a supplemental letter, dated August 12, 1987, in which he stated that he had reviewed the autopsy records, slides and hospital records of the deceased miner, in order to address a question regarding the relationship, if any, between occupational exposure and adult respiratory distress syndrome (DX 26). In sum, Dr. Bush stated: "I can find no relationship between the two conditions." In making this determination, Dr. Bush stated:

> Adult respiratory distress syndrome is the result of an <u>acute</u> injury to the lung, in this case, resulting from shock developing secondary to an acute myocardial infarction. The adult respiratory distress syndrome patient typically responds well to treatment of the acute injury but after several days respiratory symptoms develop. This was the case with Mr. Keilman. Typically, death results in 10 to 40 days from the onset of the respiratory symptoms in spite of measures to correct the disorder and support the respiratory system. This, too, is the clinical history of Mr. Keilman's hospitalization.
>
> Because of the mild degree and limited extent of the coal-workers' pneumoconiosis, I cannot conclude that it contributed in anyway to the inevitable death of this patient.

(DX 26).

Dr. Joshua A. Perper, who is Board-certified in Anatomical and Forensic Pathology and also has a law degree (DX 9), issued a lengthy report, dated May 10, 2000 (DX 10), in which he summarized various reported smoking and occupational histories, as well as the available medical evidence. Furthermore, Dr. Perper provided his own findings on microscopic examination of the autopsy tissue, and answered various questions. In addition, Dr. Perper included some references to medical literature, including an appendix which discussed "Coal workers' pneumoconiosis and associated centri-lobular emphysema." In summary, Dr. Perper concluded:

> 1. Mr. Keilman had evidence of mild simple coal workers' pneumoconiosis associated with centrilobular emphysema.
>
> 2. Mr. Keilman's simple coal workers' pneumoconiosis was a result of occupational exposure of thirty-four (34) years of work as a coal miner.
>
> 3. Simple coal worker's pneumoconiosis with associated emphysema and chronic lung disease was a substantial contributory cause of Mr. Keilman's death. The mechanisms of death was through both direct and indirect through hypoxemia (sic), the precipitation of an acute myocardial infarction, and an increase (sic) susceptibility to an acute bronchopneumonia, that complicated the myocardial infarction.

(DX 10).

Dr. Erika C. Crouch, who is Board-certified in Anatomic Pathology (DX 12), issued a pulmonary pathology consultation report, dated September 25, 2000 (DX 13). Dr. Crouch listed various medical data which she had reviewed. Furthermore, Dr. Crouch set forth her own microscopic findings on examination of the autopsy slides. Based upon the foregoing, Dr. Crouch stated:

> Diagnosis:
>
> | | | |
> |---|---|---|
> | Lungs, autopsy | -: | coal dust deposition and changes consistent with mild, simple coal workers' pneumoconiosis (see Comment) |
> | | -: | organizing diffuse alveolar damage and pneumonia |
> | | -: | emphysema |
>
> Comment:
>
> The slides show coal dust deposition and some changes that suggest coal workers' pneumoconiosis. However, only three slides are available for review, and review of the autopsy report suggests that there were a total of thirty autopsy slides including nine of lung tissue. Although the pathologic findings evident on the three available slides are consistent with mild simple coal workers'

> pneumoconiosis, a definitive assessment of severity cannot be provided based on the available materials. If the changes are representative of the lung as a whole, it is my opinion that occupational coal dust exposure could not have caused a clinically significant degree of functional impairment or disability and could not have caused or otherwise hastened this patient's death from myocardial infarction.

(DX 13).

Dr. Bush issued a supplemental report, dated October 5, 2000 (DX 14), in which he reviewed the autopsy protocol, death certificate, and 27 histologic slides. Based upon the foregoing, Dr. Bush concluded:

> The pathologic findings suggest that cause of death is adult respiratory distress syndrome resulting from hypotension due to an extensive myocardial infarction. Other evidence for hypotension of significant degree is the presence of early acute tubular necrosis in the kidneys (section O) and marked cerebral edema.
>
> The coalworkers' pneumoconiosis is mild and so limited in degree and extent that it could have played no role in death from hypotensive effects on the lung, kidneys and brain. The coalworkers' disease was so mild and limited in extent that death would have resulted at the same time and in the same manner if Mr. Keilman had never been exposed to coal mine dust.
>
> My review of the histologic slides leads me to the same conclusions I reached when I originally performed the autopsy thirteen (13) years ago.

(DX 14).

Dr. Richard L. Naeye, who is Board-certified in Anatomic and Clinical Pathology, issued a report, dated October 17, 2000 (DX 23). Dr. Naeye reviewed copies of the death certificate, autopsy report, and other medical information, including some clinical test results. Furthermore, Dr. Naeye noted an underground coal mine employment history of 33 or 34 years; and, conflicting cigarette smoking histories of 1.5 packs/day for 6 years and 0.5 packs/day for 16 years. Moreover, Dr. Naeye received 27 glass slides from the miner's autopsy, 6 with lung tissue and 4 with heart tissue. In addition, Dr. Naeye analyzed Dr. Perper's opinion. While agreeing with Dr. Perper that the infarct in Mr. Keilman's heart took place 4 weeks before he died, Dr. Naeye disagreed with Dr. Perper's conclusion regarding the role of pneumoconiosis in the miner's death. In criticizing Dr. Perper's opinion, Dr. Naeye noted the following: Dr. Perper did not recognize that the miner had clinical evidence 3-4 years before he left the mines; Dr. Perper apparently is under the misconception that the only type of coronary artery disease which leads to myocardial insufficiency and infarcts is arteriosclerosis in the large coronary arteries; Dr. Perper ignored the fact that the miner did not have any recognizable abnormalities in lung function several after leaving the coal mines; *simple* pneumoconiosis does not advance after cessation of coal mine employment; and, citing medical literature, Dr. Naeye stated: "Airway obstruction caused by

centrilobular emphysema and bronchitis that is severe enough to preclude a miner from working is very rare if indeed it occurs at all in the absence of smoking or complicated CWP." In conclusion, Dr. Naeye stated:

> In summary, this man had been disabled for years before his death by the consequences of a herniated disc and coronary artery disease. His CWP was far, too mild to have have (sic) caused any measurable abnormalities in lung function, any disability or to have hastened his death.

(DX 23).

Dr. Crouch issued a supplemental report, dated November 20, 2002, following her receipt of additional autopsy tissue slides (*i.e.*, 30 slides, instead of only 3), as well as other medical data, including the autopsy report, death certificate, miscellaneous medical and occupational records, and, in particular, pathology reports by Drs. Perper and Naeye, and, Dr. Bush's follow-up report (DX 25). Based upon the foregoing, Dr. Crouch set forth the following diagnoses and comment:

> Diagnosis:
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> | | | |
> |---|---|---|
> | Lungs, autopsy | -: | coal dust deposition and changes consistent with mild, simple coal workers' pneumoconiosis |
> | | -: | organizing diffuse alveolar damage and pneumonia |
> | | -: | emphysema |
> | | -: | organizing peripheral thromboemboli |
>
> Comment:
>
> The available autopsy materials are sufficient to evaluate the lung for the presence or absence of occupational lung disease. It is also in my opinion that the claimant had a very mild degree of simple coal workers' pneumoconiosis as characterized by coal dust macules and micronodules. There is no evidence of massive fibrosis or complicated silicosis.
>
> Based on the findings of extensive myocardial ischemic damage, it is reasonable to conclude that the patient died secondary to the complications of myocardial infarction. In my opinion, occupational dust exposure played no role in this patient's death and would not have caused any clinically significant degree of prior respiratory impairment. Coal dust exposure does not increase risk of myocardial infarction of the left ventricle secondary to atherosclerotic cardiovascular disease. The findings reported in the autopsy report reveal no significant right ventricular abnormality to suggest associated right heart disease or cor pulmonale.
>
> In my opinion, the claimant wold have died at the same date and time and in the

> same manner whether or not he had ever been exposed to coal dust. Thus, I am in agreement with the major conclusions rendered by Drs. Bush and Naeye, and disagree with Dr. Perper.

(DX 25).

Dr. Joseph F. Tomashefski, Jr., who is Board-certified in Anatomic and Clinical Pathology, issued a report, dated December 15, 2000 (DX 28). Dr. Tomashefski cited the available medical evidence, and set forth his own findings on examination of the autopsy slides. In summary, Dr. Tomashefski stated, in pertinent part:

> Based on my review of the medical records, autopsy report, and slides prepared from Mr. Keilman's autopsy, it is my opinion, within reasonable medical certainty, that he experienced a myocardial infarct, which is the underlying cause of death.
>
> The major finding in his lungs is diffuse alveolar damage in the fibroproliferative phase. Diffuse alveolar damage is the histologic counterpart of the adult respiratory distress syndrome (ARDS), the cause of which is, in Mr. Keilman's case, most likely cardiogenic shock. Diffuse alveolar damage/ARDS constitutes an important contributory cause of Mr. Keilman's death. Other findings in Mr. Keilman's lungs include apical fibrobullous disease and focal acute bronchopneumonia.
>
> Based on the presence of a small number of coal macules and micronodules, it is also my opinion, within reasonable medical certainty, that Mr. Keilman did have minimal, simple coalworkers' pneumoconiosis. In my opinion, Mr. Keilman's simple coalworkers' pneumoconiosis is of such a mild degree that it would not have caused him any respiratory impairment or respiratory symptoms, nor was it a cause or contributory factor in his death. Since Mr. Keilman's right ventricle is of normal thickness, it is also my opinion that he did not have cor pulmonale.
>
> Neither coalworkers' pneumoconiosis, coal dust exposure, nor coal mine employment is a cause of myocardial infarction or diffuse alveolar damage. It is therefore also my opinion, within reasonable medial certainty, that Mr. Keilman's death was not related to his work in the coal mines. In my opinion, he would have died in the same manner and at approximately the same time whether or not he had ever worked in the coal mining industry.

(DX 28).

Dr. Larry E. Hurwitz, who is Board-certified in Internal Medicine and Cardiovascular Disease, issued a report, dated December 19, 2000 (DX 29). Dr. Hurwitz reviewed various records; cited a 34-year coal mine employment history ending in September 1981; a somewhat inflated smoking history ranging from ½ to 1 ½ packs per day since age 18; complaints of exertional dyspnea since 1978; complaints of exertional chest pain in February 1982; a history of hyperlipidemia involving elevation of triglycerides and cholesterol, and low HDL syndrome;

arterial blood gases which were within normal limits and responded appropriately to exercise; onset of chest pain, at home, on April 1, 1987; hospitalization from April 1, 1987 to April 27, 1987, when he died; the hospital course characterized by progressive ventricular dysfunction, pulmonary distress, and ultimate cardiac arrest from which he was not successfully resuscitated; and, findings on autopsy. Based upon the foregoing, Dr. Hurwitz stated:

> In my opinion, with reasonable medical certainty, that Mr. Keilman died of the complications of acute myocardial infarction. The complications consisted of cardiogenic shock resulting in multi-organ system failure involving the cardiac, pulmonary, and renal systems.
>
> It is my opinion that the presence of mild simple coal worker's pneumoconiosis was not a contributing factor to Mr. Keilman's death. The evidence of cardiogenic shock and poor organ perfusion was the result of the acute myocardial infarction. Mr. Keilman's exposure to coal dust had no relationship in the development or the pathogenesis of the coronary disease.
>
> I would comment that Dr. Perper noted that it was his opinion that chronic hypoxia played a role in the development of the underlying heart disease. Dr. Perper clearly ignores the fact that over 85% of all myocardial infarctions occur in coronary vessels that have less than 75% coronary obstruction and that approximately 60% of all such infarctions occur in vessels with less than 50% of obstruction. The fact that no significant atherosclerosis was identified at autopsy is hardly surprising since the vast majority of clots undergo lysis within days of the acute myocardial infarction.
>
> In conclusion, it is my opinion that the occupational exposure to respirable coal dust neither caused nor accelerated, nor contributed to Mr. Keilman's heart disease or his death. There was no evidence of cor pulmonale present either in a clinical setting nor were the autopsy findings suggestive of a diagnosis of cor pulmonary (sic).

(DX 29).

Dr. Bush issued a supplemental report, dated January 5, 2001, in which he reviewed various medical records and responded to questions posed by Employer's counsel (DX 30). In summary, Dr. Bush stated: 1. There is sufficient objective evidence data upon which to evaluate the presence or absence of occupational pulmonary disease. 2. Mr. Keilman had a mild degree of simple coal worker's pneumoconiosis at the time of his death. 3. Death resulted from acute myocardial infarction which necessitated hospitalization on April 1, 1000, and was complicated by the development of adult respiratory distress syndrome. 4. There was no causal relationship between the mild degree of simple coal worker's pneumoconiosis or coal mine dust exposure and death from myocardial infarction and consequent adult respiratory distress syndrome. 5. Mr.

Keilman would have died at the same date, time, and manner if he had never been exposed to the occupational pulmonary hazards of coal mining employment. 6. Mr. Keilman had no evidence of cor pulmonale, as evidenced by the normal heart weight and the absence of right ventricular wall thickening. In addition to the foregoing, Dr. Bush analyzed and criticized Dr. Perper's opinion, as follows:

> The report of Dr. Perper merits specific comments. On page 13, Dr. Perper takes issue with the diagnosis of adult respiratory distress syndrome because of the absence of hyaline membranes. Adult respiratory distress syndrome in its early stages produces hyaline membranes with air spaces and edema. If the patient survives, the edema fluid undergoes organization in the alveoli and interstitium, as is the case wit the lungs of Mr. Keilman. The report of Dr. Crouch clearly indicates her familiarity with this change. The histologic findings are consistent with the gross appearance of the lungs which were extremely heavy and firm, the typical appearance of lungs affected by adult respiratory distress syndrome. The clinical events in the hospital are classic for adult respiratory distress syndrome, including the presence of shock and respiratory difficulty which is progressive and frequently terminates in death despite measures to maintain respiration with tracheostomy and positive pressure ventilation techniques. These heroic clinical efforts to maintain respiration did produce some over-expansion of air spaces causing blebs visible in some areas of the lungs at autopsy.
>
> Dr. Perper chooses to define the pathologic process in the myocardium as something other than acute myocardial infarction undergoing early stages of repair as noted in my autopsy report and in my letter to Mr. Creany (08/12/87). In fact, acute myocardial infarction undergoes a continuum of pathologic change first visible about 24 hours after the infarction and processing over some weeks of time to scar formation. Defining the process as "organizing" is an option and Dr. Perper's preference does not differ with the original and current diagnosis and interpretation made by me. I also note that on page 17, Dr. Perper concludes that acute myocardial infarction was the mechanism of death.
>
> Dr. Perper finds evidence of hypoxemia that contributed to death, but a review of the records shows no evidence that hypoxemia from coal worker's pneumoconiosis or chronic lung disease was present. The most recent lifetime pulmonary evaluation by Dr. Katter shows no evidence whatsoever of hypoxemia. Hypoxemia occurred as a result of and is not the cause of myocardial infarction and hypotension with subsequent adult respiratory distress syndrome. Hypoxemia is not a requisite for the development of myocardial infarction. This is succinctly indicated in an authoritative pathology text: "The term *myocardial infarction* means necrosis of myocytes caused by a reduction or cessation in coronary artery blood flow.."..... (Medical Citation omitted).

> In attempting to fully understand the mechanisms that led to death, theorizing the presence of hypoxemia is less helpful than considering the possible effect of the recent surgery, identified as stapled surgical incision in the left upper quadrant at the time of autopsy. Unfortunately, the Lee Hospital medical records are not available at this time to clarify events leading up to the final admission to the hospital.
>
> Dr. Perper appears to exaggerate the degree of coal worker's pneumoconiosis and centrilobular emphysema (mild) and theorizes the occurrence of hypoxemia from unclear mechanisms which are well beyond reasonable medical certainty.

(DX 30).

Dr. Tomashefski issued a supplemental report, dated January 9, 2001 (DX 31), in which he reviewed the medical records from Lee Hospital regarding Mr. Keilman's treatment from April 1, 1987 until his death on April 27, 1987. In summary, Dr. Tomashefski stated:

> My review of the records of Mr. Keilman's final hospitalization supports my previous opinion that Mr. Keilman had experienced a massive myocardial infarct complicated by cardiogenic shock and adult respiratory distress syndrome. The autopsy findings described in my previous report are compatible with a subacute myocardial infarct of 26 days' duration, and with the fibroproliferative phase of diffuse alveolar damage of approximately the same age.
>
> It is still my opinion that the underlying cause of Mr. Keilman's death is a myocardial infarct. ARDS secondary to the cardiogenic shock is an important contributory cause of Mr. Keilman's death.
>
> My review of these records does not change my opinion, expressed in my previous report, that Mr. Keilman had minimal simple coalworkers' pneumoconiosis which either caused nor contributed to his death.

(DX 31).

Dr. Gregory J. Fino, a B-reader who is Board-certified in Internal Medicine and Pulmonary Disease, issued a detailed report, dated January 29, 2001, in which he reviewed the available medical evidence (DX 32). At the end of his well-documented report, Dr. Fino set forth the following conclusions:

1. Simple coal workers' pneumoconiosis was present pathologically.

2. Prior to this man's death, there was no clinical indication of simple coal workers' pneumoconiosis.

3. Simple pneumoconiosis neither caused nor contributed to any disability.

4. This man's death was due to a myocardial infarction with subsequent adult respiratory distress syndrome.

5. Neither simple coal workers' pneumoconiosis nor any other type of respiratory disease caused, contributed to, or hastened this man's death.

6. This man would have died a and when he did and in the same manner, that is, due to a heart attack, had he never stepped foot in the mines.

(DX 32).

Dr. Waheeb M. Rizkalla, who is Board-certified in Anatomic and Clinical Pathology (CX 2), an Associate in Pathology, issued a report, on or about January 23, 2002, entitled "Autopsy Protocol," in which he is listed as "Reviewer." (CX 1). Attached to the report is a list "Materials Reviewed," which primarily includes the available medical data, and also refers to "Employment history and records." Dr. Rizkalla's "Autopsy Protocol" sets forth a clinical history and microscopic description. Based upon the foregoing, Dr. Rizkalla set forth the following final anatomic diagnoses:

Subacute Myocardial Infarction, Very recent, with granulation Tissue and
Chronic Mononuclear Inflammatory Cell Infiltrate

Simple Coal Worker's Pneumoconiosis, Moderate
Centrilobular Emphysema
Acute Bronchopneumonia with Organization and Diffuse Alveolar Damage

(CX 1).

In addition, Dr. Rizkalla reported the following clinicopathological summary:

This 57-year-old, white male died from myocardial infarction (at different stages). Autopsy revealed simple coal worker's pneumoconiosis complicated by broncho-pneumonia with organization and centrilobular emphysema. Coal worker's pneumoconiosis is considered a substantial contributing factor in his death.

(CX-1).

In Dr. Rizkalla's cover letter/report, dated January 25, 2002 (CX 1), he cited the medical records and microscopic slides, and responded to various questions. Except for the introductory paragraph, the following is the full text of Dr. Rizkalla's report:

Mr. Keilman had simple coal worker's pneumoconiosis of a moderate degree with

centrilobular emphysema. Considering his occupational history of working in the coal mining industry for more than thirty years, the clinical evaluations, including chest x-rays and ventilatory studies and the autopsy information, there is unequivocal evidence that Mr. Keilman developed simple coal worker's pneumoconiosis during his life.

Coal worker's pneumoconiosis was a substantial contributing factor in his death. Mr. Keilman died from acute myocardial insult or injury in spite of minimally-affected coronary vessels as described by the prosector. Acute myocardial injury occurs when the amount of the blood decreases or the quality of the blood will be less than optimum to meet the demand of the myocardium.

The decedent had chronic obstructive pulmonary disease with abnormal function a few years prior to his death. He had abnormal respiratory functions with the diagnosis of restrictive and obstructive pulmonary disease which affects the oxygenation of blood (inducing hypoxia) which will be a delineating factor in developing terminal myocardial injury. When the patient developed the myocardial injury subsequent hypotension occurred and lung injury in the form of diffuse alveolar damage (adult respiratory distress syndrome or shock lung).

To summarize, Mr. Keilman had simple coal worker's pneumoconiosis of moderate degree with centrilobular emphysema and no significant atherosclerotic coronary artery disease affecting either the major coronary vessels or the small blood vessels in the myocardium.

Mr. Keilman quit smoking many years prior to his death. It is unlikely that the smoking played any role in the mechanism of his death. He had no other disease process that would add to or explain the mechanism of myocardial infarction sustained prior to his death except the lung disease.

(CX 1).

Dr. Bush issued another supplemental report, dated February 22, 2002, in which he reviewed three additional histologic slides (EX 1). Dr. Bush stated, in pertinent part:

These lung slides show evidence of changes consistent with adult respiratory distress syndrome including organizing interstitial and intra alveolar edema, sloughing and hypertrophy of alveolar lining cells and interstitial fibrosis. Areas of localized acute bronchopneumonia with micro-abscess formation are noted, as well.

The histologic slides also show a mild degree of simple coal worker's pneumoconiosis manifested by coal worker's micronodules which are less than 0.2

> cm and average 0.1 cm. One histologic slide contains no micronodules, one contains one and one slide contains five micronodules. Two of the slides contain a moderate degree of centrilobular emphysema unrelated to dust pigment. These additional slides show essentially identical findings to those described in my report of 10/05/00 and the original autopsy report of 04/28/87, both prepared after examining the histologic slides from the autopsy.

(EX 1).

Dr. Hurwitz, whose findings were initially outlined in a report, dated December 19, 2000 (DX 29), as summarized above, testified at deposition on January 31, 2002 (EX 2). Dr. Hurwitz clarified the miner's smoking history somewhat, noting that, although Mr. Keilman had a prior cigarette smoking history, "fortunately he had not continued to do (it) for a number of years." (EX 2, p. 13).[3] The crux of Dr. Hurwitz's testimony, however, was to reiterate his conclusion that Mr. Keilman died a cardiovascular death; namely, a myocardial infarction and complications therefrom, in particular, cardiogenic shock; and, restate his opinion that pneumoconiosis, centrilobular emphysema, or any pulmonary condition in no way affected the miner's clinical course (EX 2, pp. 12-20, 28-29). Dr. Hurwitz, a Board-certified cardiologist, who has specialized in that field since 1973 (EX 2, p. 10) acknowledged that he is not a pathologist or pulmonologist (EX 2, p. 27). Although Dr. Hurwitz deferred to the pathologic and pulmonary findings of the experts in those fields, from a cardiovascular standpoint, he essentially agreed with most of the pulmonologists and/or pathologists who attributed the miner's death to a heart-related problem, unrelated to pneumoconiosis, such as Drs. Fino, Bush, Tomashefski, and Naeye. On the other hand, Dr. Hurwitz had "trouble trying to understand" the basis for Dr. Perper's conclusions. Furthermore, Dr. Hurwitz analyzed the underlying rationale for Dr. Perper's conclusion, and stated that Dr. Perper's opinion "that his (Mr. Keilman's) pulmonary disease was responsible for the occurrence of the myocardial infarction...is completely unfounded (EX 2, pp. 21-25). In addition, Dr. Hurwitz briefly discussed Dr. Rizkalla's opinion, and stated, in pertinent part, that he agreed with those parts of Dr. Rizkalla's comments which indicate that Mr. Keilman suffered a significant myocardial infarction and adult respiratory distress syndrome. However, Dr. Hurwitz expressly disagreed with Dr. Rizkalla's opinion that the miner's pneumoconiosis and centrilobular emphysema played a role in the miner's death (EX 2, pp. 25-26).

Dr. Bush, the prosector and Board-certified pathologist, who issued multiple reports, as outlined above, testified at deposition on February 7, 2002 (EX 3). In summary, Dr. Bush described the cause of death as follows:

> Mr. Keilman had died as a result of a condition called adult respiratory distress

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[3] The December 19, 2000 report indicates a cigarette smoking history of ½ to 1 ½ packs per day "from age 18" (DX 29). This suggests that Dr. Hurwitz had previously thought that Mr. Keilman continued to smoke until his death.

> syndrome, which ended in total respiratory failure, pursuant to a hospital admission several weeks before for a massive myocardial infarction that caused a fairly prolonged period of hypotension shock.

(EX 3, p. 15). Furthermore, Dr. Bush reiterated his prior diagnosis of a mild degree of simple coal workers' pneumoconiosis on autopsy; and, stated that this is, in essence, an incidental finding, which would not have interfered in any way with the miner's lung function during his lifetime (EX 3, pp. 19-21). Dr. Bush testified that his opinion regarding the issues of pneumoconiosis and death were essentially in agreement with those of Drs. Fino, Tomashefski, Hurwitz, Crouch, and Naeye (EX 3, p. 22). On the other hand, Dr. Bush disagreed with the opinions of Drs. Perper and Rizkalla regarding the role of pneumoconiosis in the miner's death. Dr. Bush, and most of the other physicians of record, opined that the miner's heart attack caused the shock condition which, in turn, damaged the lungs and caused hypoxemia. Dr. Bush disagreed with the opinions of Drs. Perper and Rizkalla, who related the miner's heart attack, hypoxemia, and death to underlying chronic lung diseases, such as pneumoconiosis and centrilobular emphysema, while citing Dr. Katter's 1984 examination of the miner showed no evidence of hypoxemia, pulmonary function studies described by Dr. Pickerill as showing a mild restrictive disease, and correlating them with the autopsy findings (EX 3, pp. 22-27, 38-39, 50-51).

Dr. Fino, whose opinion was initially set forth in a report, dated January 29, 2001 (DX 32), as outlined above, testified at deposition on February 13, 2002 (EX 4). Dr. Fino noted objective testing on lung function which was normal in 1982 and 1984, and cited the absence of any respiratory complaints in primary care records from January 11, 1985 through December 12, 1986, which indicated no respiratory impairment (EX 4, pp. 13-16). Dr. Fino testified that during the last month of the miner's life (*i.e.,* April 1987), Mr. Keilman suffered a large heart attack, which induced cardiogenic shock. The cardiogenic shock caused adult respiratory distress syndrome, and resulted in complications, and subsequently death (EX 4, pp. 16-18). Dr. Fino acknowledged that, if Mr. Keilman had actually suffered from clinically significant emphysema and/or pneumoconiosis, which caused more than a mild abnormality on lung function, it possibly could change his overall opinion (EX 4, pp. 33-34). However, the crux of Dr. Fino's opinion was that although the miner had evidence of the disease pathologically, it was not clinically significant. Accordingly, Dr. Fino reiterated that the miner "would have died when he in the same manner had he never been exposed to coal mine dust, due to a heart attack leading to adult respiratory distress syndrome." (EX 4, pp. 19-20).

Discussion and Applicable Law

As set forth above, the Employer stipulated, and I find, that Mr. Keilman had simple pneumoconiosis, which arose from his 33+ years of coal mine employment. However, in order to be eligible for benefits, Claimant must also establish that the miner's death was due to

pneumoconiosis, as provided in the Act and applicable regulations.

Death due to Pneumoconiosis

Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c), as amended, which states, in pertinent part:

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at § 718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.
>
> (5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

As outlined above, the death certificate states that the miner died of a myocardial infarction, but also lists adult respiratory distress syndrome and chronic pulmonary disease as significant conditions contributing to death, albeit not to the immediate cause (DX 7). However, Dr. Patel, who signed the death certificate, failed to specify whether the above-referred respiratory and/or pulmonary conditions were related to coal mine employment. Furthermore, Dr. Patel did not set forth the basis for his conclusions, or even indicate whether he considered the autopsy findings. Therefore, I accord little weight to the miner's death certificate. Moreover, Dr. Patel's qualifications are not of record.

The crux of this case rests with the relative weight to be accorded the opinions of Drs. Bush (DX 11, 14, 26, 30; EX 1,3), Perper (DX 10), Crouch (DX 13, 25), Naeye (DX 23), Tomashefski (DX 28, 31), Hurwitz (DX 29; EX 2), Rizkalla (CX 1), and Fino (DX 32; EX 4). Of the foregoing, only Dr. Perper and Dr. Rizkalla found that the miner's coal worker's

pneumoconiosis was a substantially contributing factor in the miner's death. In contrast, Drs. Bush, Crouch, Naeye, Tomashefski, Hurwitz, and Fino found that the miner's relatively mild simple coal worker's pneumoconiosis was essentially an incidental pathology finding, which did not cause, substantially contribute, or hasten the miner's death.

All of the foregoing opinions, including those by Drs. Perper and Rizkalla, appear, on their face, to be well-reasoned and documented. However, the burden rests with the Claimant to establish the elements of entitlement, including "death due to pneumoconiosis," by a preponderance of the evidence. Based upon my analysis of the record, in particular, the above-listed medical opinions, I find that the Claimant has clearly failed to meet her burden of proof.

In making this determination, I first note the numerical superiority of those physicians who found the miner's death was not due to pneumoconiosis. More importantly, the record overwhelmingly establishes that the miner suffered a massive myocardial infarction within a month of his death, and, adult respiratory distress syndrome, and other complications resulting therefrom. Accordingly, I find that the opinions of physicians with expertise in cardiology and/or pulmonary medicine are most important. In the present case, the opinions of the Board-certified pathologists (*i.e.*, Drs. Bush, Crouch, Naeye, Tomashefski, Perper and Rizkalla) are conflicting. However, Dr. Hurwitz, a Board-certified cardiologist, and Dr. Fino, a Board-certified pulmonary specialist, both found that the miner's death was unrelated to pneumoconiosis. Moreover, Dr. Hurwitz expressly disagreed with the conclusions of Drs. Perper and Rizkalla, and, provided extensive testimony why Dr. Perper's opinion, in particular, was, in fact, poorly reasoned. Similar criticism of Dr. Perper's opinion was set forth by other physicians, such as Dr. Bush.

In view of the foregoing, I credit the opinions of Drs. Hurwitz and Fino, Board-certified specialists in cardiology and pulmonary medicine, respectively, as buttressed by the opinions of Drs. Bush, Crouch, Naeye, and Tomashefski, over the opinions of Drs. Perper and Rizkalla. Accordingly, I find that the Claimant has failed to establish death due to pneumoconiosis under § 718.205(c), or by any other means.

Conclusion

Although the evidence shows that the miner had simple pneumoconiosis which arose from his 33+ years of coal mine employment, it does not establish that pneumoconiosis caused, substantially contributed to, or hastened the miner's death. Therefore, I find that the Claimant is not entitled to benefits under the Act and applicable regulations.

Attorney's Fees

The award of an attorney's fee under the Act is permitted only in the cases in which Claimant is found to be entitled to benefits. Since benefits are not awarded in this case, the Act

prohibits the charging of any fee to the claimant for services rendered to him in pursuit of this claim

ORDER

It is ordered that the claim of Irene G. Keilman, surviving spouse of Wilbert A. Keilman, Sr., for black lung benefits under the Act is hereby **DENIED**.

RICHARD A. MORGAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. 725.481, any party dissatisfied with this Decision and Order may appeal to the Benefits Review Board within 30 days from the date of this Decision and Order, by filing a notice of appeal with the ***Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601.*** A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.